**Testimony on Alaska House Bill 63**
**For the Alaska State Legislature**
**House Labor and Commerce Committee**

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Good afternoon. My name is Sandra Steingraber. I am a Ph.D. biologist with a background in systems ecology. For the past 15 years, I've been researching chemical contaminants with the power to enter the human body and, at low levels, tinker with our cellular machinery in ways that raise the risk for health problems—by turning on and off genetic switches, for example, or by disrupting hormonal signals that guide our development, or by interfering with the wiring together of brain cells during infancy and childhood.

I'm the author of three books on the topic of environmental health, including *Living Downstream: An Ecologist's Personal Investigation of Cancer and the Environment*, which has recently been adapted for film. This documentary is screening tonight here in Anchorage. Yesterday, I had the opportunity to address the Alaska Academy of Family Physicians on the topic of environmental health threats, including brominated flame retardants, at its annual meeting in Girdwood, which is what brings me from upstate New York—I'm on the faculty at Ithaca College—to Alaska. I have previously provided Congressional briefings on environmental health, testified before the President's Cancer Panel, and spoken in the European Parliament.

I am here to urge support for Alaska House Bill 63, which seeks to prohibit the sale and distribution of one uncommonly dangerous substance, the brominated flame retardant known as decaBDE.

As science advisor to the California Breast Cancer Research Program, I had the opportunity to review the literature on the health effects of polybrominated diphenyl ethers as they are relevant to breast cancer risk. My chapter on PBDEs appears in the recently released final report, *Identifying Gaps in Breast Cancer Research* (http://www.cbcrp.org/sri/reports/identifyingGaps/SRI_FLAME_083107.pdf.) I include excerpts from this chapter in my written testimony, which I'm pleased to s submit to you.

Here now are a few highlights of that report. First, brominated flame retardants

are very stable molecules, which means they are remarkably persistent in the environment and in human tissue. They don't break down easily. A molecule of decaDBE is built like a bicycle. It has two wheels made up of benzene rings held together by a sturdy frame of oxygen. That property of chemical stability means that brominated flame retardants are subject to long-distance transport and come down to earth in cold places. Alaskans are thus at special risk for exposure to brominated flame retardants.

If these stubborn bicycle-shaped molecules were biologically inert, these exposures might matter less. Sadly, however, they have the power to mimic hormones. One of them is estrogen. This is why we in the scientific community are concerned about their possible link to breast cancer.

And now I'd like to speak to you as mother. Recent studies have identified brominated flame retardants as developmental neurotoxicants. That means they affect not just our breasts but also our minds. They can cross the placenta during pregnancy and sabotage the architecture of the fetal brain in ways that interfere with learning and cognitive development in childhood. They are brain poisons.

Flame retardants are the new lead paint chip.

Moreover, compared to adults, children have levels of brominated diphenyl ethers in their bodies—probably because they ingest more house dust. Brominated flame retardants do not stay in the foam and plastic materials into which they are embedded. And when they seed themselves into house dust, they will find their way into the brains of our children and interfere with development.

An individual parent can do little to prevent this toxic trespass. I am the mother of two young children myself. I am a conscientious parent, but I am not a HEPA filter. A public health approach that surrounds kids with brain poisons and enlists mother and fathers to serve as security detail is as failure-prone with deca as it was with lead paint.

At the very least, learning disabilities and breast cancer are expensive problems that are diminishing our productivity and burdening our educational and health care systems. At the very most, chemicals such as deca--which enter our bodies without our consent, contribute to the diminishment of intelligence and alter hormones that we know play a role in breast cancer--are the destroyers of children and mothers alike. They violate our human right to a safe environment. Thank you, Alaska, for safeguarding families by taking up this bill.

**Appendix: Excerpt from "Polybrominated Flame Retardants" in *Identifying Gaps in Breast Cancer Research* (Oakland, California: California Breast**

**Cancer Research Program).**

## Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of persistent halogenated organic compounds widely used as flame retardants. Like dioxins and PCBs, PBDE molecules resemble bicycles. They consist of two phenyl rings studded with bromine atoms (the wheels) and attached by an oxygen bridge (the frame). When PBDE molecules are exposed to heat—as in a house fire—the bromines detach and quench the flames.

PBDEs, like PCBs, exist as more than 200 potential congeners. However, only three mixtures have been available for commercial use, as identified by the average number of bromines in the dominant congener: Deca, Octa, and Penta. Deca, with ten bromine atoms, is used in hard polystyrene plastics, textiles, and electronic equipment such as televisions. It is also used in polyethylene for wires, cables, and pipes. Octa- PBDE, with eight bromine atoms, has primarily been used in the plastic housings of computer monitors and in circuit boards. With five bromine atoms, Penta-BDE has been used in flexible foam products, such as polyurethane furniture cushions, carpet padding, and mattresses. Penta has also been used in rigid foams.[1-4] These three mixtures are not strictly homogeneous and can contain PBDEs with other numbers of bromine; for example, Penta can contain some fraction of Tetra-BDE. The only mixture currently available in the United States and the European Union is Deca-BDE. The European Union banned Octa and Penta in 2004, and the sole U.S. manufacturer voluntarily stopped production in the same year.[5]

PBDEs first became commercially available as flame retardants in 1960[4] and have been widely used throughout the world for the last 30 years. Usage has tripled during the previous two decades.[6] In 2001, approximately 67,440 metric tons of PBDEs were manufactured, with the majority of use occurring in North America.[7] The U.S. has been, by far, the predominant producer and user of Penta.[8] Over time and under ordinary conditions of use, PBDEs have diffused out of the polymer matrices in which they were embedded and are now a ubiquitous contaminant of indoor and outdoor environments.[3, 8-10] By the late 1990s, Swedish researchers had documented exponential increases in PBDE levels in breast milk samples collected from 1972 to 1997. These findings were one factor that inspired a ban on PBDE manufacture in the European Union. Between 1998 and 2002, levels in human milk in Sweden decreased significantly.[3, 4, 8, 10]

Here in the United States, PBDE levels in Great Lakes fish rose rapidly during the 1980s and 1990s and doubled in less than three years. PBDEs have also turned up in commonly consumed fish, including salmon, mackerel, swordfish, herring, catfish, and shellfish; and they have been detected in many types of wildlife around the world, with some of the highest levels found in harbor seals in the San Francisco Bay.[4] This discovery, together with the Swedish breast milk results, prompted researchers to measure levels of PBDEs among U.S. human residents. U.S. inhabitants have the highest documented levels of PBDEs in the world. These levels are 10- to 100-fold higher than levels observed in Europe, Asia, or New Zealand.[7, 8, 11-13] Moreover, as seen in fish and wildlife, body burdens appear to be increasing.[4, 10, 11, 14, 15]

Owing to their similar molecular structure and toxic profile, PBDEs are often referred to as the 'PCBs of the future.'[8, 16] The less-brominated forms—Penta and Octa—are the more persistent, lipophilic, and biologically active. Some of the congeners contained in Penta-DBE have been identified as estrogenic.[4] By contrast, Deca-BDE is less well absorbed and less bioaccumulative. Its bulky size and high molecular weight restrict its toxicity and ability to biomagnify. Furthermore, Deca-BDE binds strongly to soil and sediments, limiting its bioavailability.[2, 3, 8, 10, 17] Ominously, however, debromination of Deca can generate the lighter, more toxic forms. The degree to which Deca degrades to the less brominated congeners in the environment is a source of ongoing debate. Despite their widespread use, very little is known about the human health effects of exposures to PBDEs. Only a few epidemiological investigations have been conducted.[18, 19] Limited data from animal studies suggest that these compounds may exert endocrine-disrupting effects at levels close to those being documented in the current U.S. population, especially among children,[20] making them of particular concern for breast cancer.

## PBDEs in the Environment

PBDEs are detectable in many environmental media, including air, soil, household dust, clothes dryer lint, sewage, fish, and wildlife.[1, 2, 10, 11, 13, 25-33] In North America, Penta-BDE is the primary contaminant found,[11] although Deca is often dominant in house dust. A recent meta-analysis of environmental PBDE concentrations reported exponential increases over the last 30 years, with a doubling time of approximately four to six years.[11] This study also demonstrated especially high levels of contamination in North America compared to Europe and Japan, the other two regions of the world with available data.[3, 11, 13, 29]

Sources of contamination have not been fully evaluated. One important non-point source of contamination is thought to be household trash, which often contains furniture, bedding, foam cushions, and electronics loaded with PBDEs. No information, however, is currently available on the degree to which incineration and landfills contribute to environmental contamination.[8] Recent work in Great Britain along urban-rural transects suggests that cities themselves may be sources, possibly from leakage of PBDEs from indoor to outdoor air.[34] Because incomplete combustion may produce brominated dioxins and furans, concern has also focused on incomplete incineration and accidental fires as additional sources of exposure.[8, 10] Sewage sludge is a well- documented source of persistent environmental contamination, especially for Deca, which binds strongly to sediment.[11, 29] Concentrations of PBDEs in water generally haven't been assessed due to their low solubility in water.[8, 29] Fish and marine mammals tend to have higher levels than do their terrestrial counterparts.[11, 29]

## PBDEs in People

PBDEs have been detected in human blood, breast milk, umbilical cord blood, and in adipose, brain, liver, and placental tissue.[7, 8, 10, 12, 13, 35-40]

Over the past three decades, PBDE body burden levels have increased 100-fold, representing a doubling time of approximately five years. On average, U.S. blood levels

(35ng/g lipid, which equals 35 ppb) are 17 times higher than in those seen in European populations (2 ng/g lipid or 2 pbb).[11] PBDE levels in the breast milk of U.S. mothers are 10–100 times those seen in the breast milk of European mothers.[40] Within the U.S., human body burdens of PBDEs vary wildly. Most PBDE researchers report levels between 4 and 400 in human blood and breast milk. However, in 2005, a team of researchers found individuals in New York City with levels as high as 9,630 ppb (in a 32-year-old man) and 4,060 ppb (in a 23- year-old woman). These levels are 4 to 9.5 times higher than any previously reported in people anywhere in the world.[41, 42]

The exponential rise in body burden levels of PBDEs stands in stark contrast to the temporal trends of other well-known organohalogenated compounds, many of which have markedly declined over the last few decades.[7, 15] A recent analysis comparing body burden levels of PBDEs, dioxins, furans, and PCBs measured in current and archived sera from 1973 in a U.S. population demonstrated this dramatically changing exposure profile.[7] PCBs, dioxins, and furans all declined dramatically during the 30-year span (1973–2003) marked by the collection of the two sets of sera, presumably reflecting the banning and regulation of these compounds.

In contrast, PBDEs were virtually undetected in the 1973 samples but were the predominant compound in the current sera. On average, these levels were more than twice those of current levels of PCBs, and 100 to nearly 2,000 times those of the dioxins and dibenzofurans. These levels may decline in the U.S. population with the recent ban of Octa and Penta. Initial reports from Sweden indicate that body burden levels there may be leveling off or even declining after exponential increases observed during the 1980s and 1990s.[11] However, the Swedish ban on PBDEs is more comprehensive.

## Routes of Exposure

Routes of human exposure to PBDEs and the relative contribution of different sources depend on the congener or congener group, the country, and the life stage of the individual.[1, 8, 43, 44] Food is a vector for exposure but appears to play a lesser role than it does for other common persistent organic pollutants.[45-47] There is now good evidence that both diet and the indoor environment (probably inadvertent dust ingestion) contribute to exposure to Penta-BDE in adults in the U.S.[47] The indoor environment – both dust ingestion and dust inhalation – may dominate for exposure to Deca- BDE in the U.S.[43]

*The Debromination Question*

Some human exposure to bioactive Penta- and Octa-PBDE may come from the degradation of Deca. In contrast to industry claims, several studies now indicate that Deca can debrominate under ordinary environmental conditions, including through exposure to sunlight and via metabolism. Bacteria and fish, for example, can convert Deca into lighter brominated congeners [10, 17, 32, 48-52] and there is some evidence for metabolic debromination of Deca in mammals.[53] While Deca is not easily absorbed across the gut wall, its less brominated congeners are.[10, 11] Moreover, recent studies of workers exposed to Deca indicate that some fraction of Deca is absorbed. Deca has also been detected in blood and breast milk samples from the general population.[4]

*Occupational Exposures*

Occupational exposures may be important for workers in computer and electronic manufacturing, recycling, and disassembly plants and in PBDE formulation facilities.[1, 3, 8, 11, 54]

*Diet*

Diet is not the sole significant route of exposure to PBDEs and appears to explain only a portion of the variability in PBDE levels.[10, 35, 38, 44, 47]

Several lines of evidence suggest a smaller role for diet than the lipophilic nature of PBDEs might suggest.[12, 35, 37] First, research has established a link between Penta-BDE concentrations found in people with the quantities found in dust from their homes, independent of diet.[47] Second, although levels of fish contamination are orders of magnitude higher in North America than they are in Japan or Europe, analyses in U.S. populations tend not to see a large correlation between fish consumption and body burden levels of PBDEs.[11, 14, 47] Third, PBDE levels are not positively correlated with age. Indeed, children have higher body levels than adults.

Two studies have reported that PBDE levels in U.S. children are two to five times those found in adult populations.[36, 55] One case study of a San Francisco Bay Area family[36] found blood levels of Deca comparable to levels seen in Swedish workers manufacturing and/or dismantling Deca-treated products.[56, 57] Total PBDE levels in the children, which ranged from 151–651 ng/g lipid, approached the 95th percentile of what has been reported in U.S. adult populations.[7]

All together, these results suggest that diet is not the sole or primary route of exposure for children and adults. Ingestion of breast milk does appear to be the primary route of exposure among breast- feeding infants.[1] PBDEs can also pass through the placenta.[1] Liver tissues from seven live-born and four stillborn U.S. infants attest to prenatal PBDE transfer from mother to offspring. The mean level was 23.1 ppb in these infants, and the median 15.2 ppb, lipid.[39]

*Household Dust Ingestion and Inhalation*

Among children and adults, dust appears to be an important vector for exposure. Unlike PCBs, PBDEs are a pervasive indoor pollutant found at high levels in household and office dust.[7, 31, 32, 47] A recent analysis of PBDE levels in breast milk samples reported a strong correlation with household dust samples, and to a lesser degree, with dietary consumption of dairy and meat products.[47] Thus, inhalation and ingestion of dust may be a particularly significant route of human exposure, especially among young children.[36, 58] Allen et al.[43] found that inhalation of dust may be important for exposure of adults to Deca.

The degree to which leaching of PBDEs from products in the home or office—directly into the indoor environment or through direct dermal absorption from furniture/mattresses—contributes to human exposures requires further exploration. House

dust samples from the Washington DC area found no correlations between total PBDE concentration and year of house construction, type of flooring, presence of carpeting, or number of television sets or personal computers in the home.[32] A study focusing on house dust likewise found no direct connection between household products known to contain PBDEs and levels of PBDEs in dust.[47] However, when using x-ray fluorescence to screen for bromine, researchers in Boston did definitively link PBDE concentrations in dust with bromine concentrations of household furnishings, including TVs, power strips, CD players, VCRs, alarm clocks, chairs, couches, mattresses, pillows, and futons.[45]

## Critical Review of the Literature

In spite of the widespread usage of and documented human exposures to PBDEs, remarkably little data on the health effects of PBDEs exist. The full-bore introduction of PBDEs into electronics and furniture manufacturing in the 1970s preceded a systematic investigation of their toxicological properties. Concerns about the environmental health impacts of PBDEs were greatly heightened after documentation of an exponential rise in PBDE levels in national breast milk samples in Sweden. This report was published in 1998.[59]

Thus, research on the health impacts associated with these widespread exposures is little more than a decade old. To date, no breast cancer studies have been conducted in humans. However, the virtual absence of PBDEs in human sera prior to 1973 means that the oldest cohort of U.S. women exposed to PBDEs in infancy is now only in their 30s – too young for most to develop breast cancer. For women who are old enough to be at risk for breast cancer, PBDE exposure occurred in adulthood, not during fetal, infant, or pubertal life when the mammary gland was under development and when exposures may raise the most risk for harm. Moreover, widespread human exposure to PBDEs may not yet have exceeded the latency period for carcinogenesis. Meaningful retrospective epidemiological investigations into PBDEs as a contributor to breast cancer risk are thus decades away. The suggestion that some congeners, especially Penta, act as endocrine disruptors, nevertheless, make PBDEs of particular interest with respect to breast cancer etiology.

### *Laboratory Studies*

A number of in vitro studies have suggested potential endocrine-disrupting activity for PBDEs. PBDEs, which structurally resemble thyroid hormone, have been shown in vitro to disrupt thyroid activity by competitively binding to the T4 receptor site.[3, 60, 61] They may also bind to the plasma carrier protein transthyretin, causing more rapid metabolism of thyroid hormone.[62]

However, the resemblance between PBDEs and thyroid hormone is not the whole story. Additional studies have shown that PBDEs – or their hydroxylated and methoxylated metabolites – can bind with estrogen receptors in vitro,[63-65] while one study reported anti-androgenic activity.[66] Furthermore, PBDE metabolites disrupt cytochrome P45017 (CYP17) enzyme activity in vitro.[67] Because CYP17 catalyzes key steps in sex hormone synthesis in humans, these results may be particularly relevant to breast cancer, although

such effects have yet to be evaluated in vivo.

### *Animal Studies*

Carcinogenicity studies in animals have only been conducted for Deca-BDE, the least toxic mixture. Based on very limited bioassay data from chronic oral dose studies in rats, the U.S. Environmental Protection Agency classified Deca-BDE as a Class C (Possible Human Carcinogen). This classification, published in 1986, was based on no human data and limited evidence of carcinogenicity in rodents, specifically increased incidences of neoplastic liver nodules in male and female rates and increased incidences of hepatocellular adenomas in male rats.[68] All of the PBDE mixtures have been shown to disrupt thyroid balance in vivo, although Deca-BDE appears to be the least potent in this regard.[3]

The mechanism by which PBDEs lower thyroid levels has not been fully characterized. Furthermore, the degree to which these findings are applicable to humans, who are considered to be less sensitive to disruption of thyroid function than rodents, is not currently known.[3] Finally, the relevance of these findings to breast cancer is not known. While there have been reports of an elevated incidence of thyroid diseases among breast cancer patients, a causal link has not been established; but this is an area of growing and intense interest to breast cancer researchers.[69]

A handful of animal studies have examined the reproductive effects of PBDEs. Structural changes were observed in the ovaries of PBDE-treated female rats,[70] and sperm function decreased in male mice exposed to Deca.[71] Furthermore, a number of studies have reported delays in puberty onset in both male and female rats exposed to PBDEs.[66, 72-74]

Stoker and colleagues reported delayed puberty in male rats as well as suppressed growth of androgen-dependent tissues following a peri-pubertal exposure. This disruption appeared to indicate that PBDE was acting as an androgen receptor antagonist.[66, 74] No studies have examined the effect of PBDEs on mammary gland development.

### *Human Studies*

To date, no epidemiologic study of breast cancer and PBDE exposures has been conducted. Two small studies from Sweden, however, suggest potential carcinogenic effects in humans. In 1998, Hardell and colleagues reported a non-significant two-fold elevated risk of non-Hodgkin's lymphoma associated with adipose levels of Tetra-BDE.[75] A later study by the same research group in Sweden reported an increased risk of testicular cancer (OR = 2.5, 95% CI = 1.02–6.0) associated with maternal, but not case, sera levels of PBDEs.[76] These latter findings are particularly intriguing with regards to breast cancer, as risk for testicular cancer is thought to be at least partially mediated by pre-/peri-natal exposures to endogenous and exogenous hormone levels. The maternal blood levels in this study, however, were collected at the time of the son's diagnosis and may not reflect the *in-utero* exposures experienced by the sons from decades prior.

Two birth cohort studies have found associations between PBDE concentrations and health effects other than cancer. In a Danish- Finnish study, the concentration of PBDEs

in breast milk was significantly higher in boys with cryptorchidism (undescended testicles) than in controls.[19] A study in Taiwan found a relationship between PBDE levels in breast milk and birth outcome: higher PBDE levels were associated with lower birth weight and shorter birth length.[18]

## Conclusions

PBDE exposures to humans are pervasive and, in contrast to other PCBs and dioxins, human body burden levels are increasing, with a doubling time of about five years.[16] While recent regulatory action to restrict the use of some PBDEs may stem the extraordinary increases in exposures observed over the last three decades, human exposures are likely to continue for decades to come, because PBDEs persist and bioaccumulate in the environment. Despite known widespread exposures, the health effects remain largely unknown. Retrospective epidemiology studies to illuminate breast cancer risks are unlikely to yield insights in the near future because the widespread commercialization of PBDEs occurred only within the last thirty years.

### References

1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Polybrominated Biphenyls and Polybrominated Diphenyl Ethers. Atlanta, GA, USA: Agency for Toxic Substances and Disease Registry (ATSDR), 2004. Available at http://www.atsdr.cdc.gov/toxprofiles/tp68.pdf.

2. Environmental Working Group (EWG). Brominated Fire Retardants: Persistent Global Pollutants (Part 1). In: Environmental Working Group (EWG). In the Dust: Toxic Fire Retardants in American Homes. Washington, DC, USA: Environmental Working Group (EWG), 2004. Available at http://www.ewg.org/reports/inthedust/part1.php.

3. McDonald TA. A perspective on the potential health risks of PBDEs. Chemosphere, 2002, 46(5):745-55.

4. Snedeker S. PBDEs - Polybrominated diphenyl ethers. In: Cornell University, Sprechter Institute for Comparative Cancer Research, Program on Breast Cancer and Environmental Risk Factors. BCERF Briefs. Ithaca, NY, USA: Cornell University, 2007. Available at http://envirocancer.cornell.edu/pbde/brief.pdf and http://envirocancer.cornell.edu/pbde/BriefBib.pdf.

5. Birnbaum LS, Cohen Hubal EA. Polybrominated diphenyl ethers: a case study for using biomonitoring data to address risk assessment questions. Environ Health Perspect. 2006, 114(11):1770-5.

6. Boston University (BU), School of Public Health, Department of Environmental Health, International Joint Commission (IJC), Health Professionals Task Force. Polybrominated Diphenyl Ethers (PBDEs). Health Effects Review. 2004, Fall.

7. Schecter A, Papke O, Tung KC, Joseph J, Harris TR, Dahlgren J. Polybrominated diphenyl ether flame retardants in the U.S. population: current levels, temporal trends, and comparison with dioxins, dibenzofurans, and polychlorinated biphenyls. J Occup Environ Med. 2005, 47(3):199-211.

8. Siddiqi MA, Laessig RH, Reed KD. Polybrominated diphenyl ethers (PBDEs): new pollutants-old diseases. Clin Med Res. 2003, 1(4):281-90.

9. Environmental Working Group (EWG). Dust and Indoor Pollution (Part 3). In: Environmental Working Group (EWG). In the Dust: Toxic Fire Retardants in American Homes. Washington, DC, USA: Environmental Working Group (EWG), 2004. Available at http://www.ewg.org/reports/inthedust/part3.php.

10. Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M. Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. Environ Health Perspect. 2001, 109 Suppl 1:49-68.

11. Hites RA. Polybrominated diphenyl ethers in the environment and in people: a meta-analysis of concentrations. Environ Sci Technol. 2004, 38(4):945-56.

*California Breast Cancer Research Program*
12. Petreas M, She J, Brown FR, Winkler J, Windham G, Rogers E, Zhao G, Bhatia R, Charles MJ. High body burdens of 2,2',4,4'-tetrabromodiphenyl ether (BDE-47) in California women. Environ Health Perspect. 2003, 111(9):1175-9.
13. She J, Petreas M, Winkler J, Visita P, McKinney M, Kopec D. PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue. Chemosphere. 2002, 46(5):697-707.
14. Schecter A, Papke O, Tung KC, Staskal D, Birnbaum L. Polybrominated diphenyl ethers contamination of United States food. Environ Sci Technol. 2004, 38(20):5306-11.
15. Sjodin A, Jones RS, Focant JF, Lapeza C, Wang RY, McGahee EE 3rd, Zhang Y, Turner WE, Slazyk B, Needham LL, Patterson DG Jr. Retrospective time-trend study of polybrominated diphenyl ether and polybrominated and polychlorinated biphenyl levels in human serum from the United States. Environ Health Perspect. 2004, 112(6):654-8.
16. Madsen T, Lee S, Olle T. Growing Threats: Toxic Flame Retardants and Children's Health. Los Angeles, CA, USA: Environment California Research and Policy Center, 2003. Available at http://www.environmentcalifornia.org/uploads/_B/5K/_B5KW8OvzEtND0dzkmq-KA/Growing_Threats.pdf.
17. La Guardia MJ, Hale RC, Harvey E. Detailed polybrominated diphenyl ether (PBDE) congener composition of the widely used penta-, octa-, and deca-PBDE technical flame-retardant mixtures. Environ Sci Technol. 2006, 40(20):6247-54.
18. Chao HR, Wang SL, Lee WJ, Wang YF, Papke O. Levels of polybrominated diphenyl ethers (PBDEs) in breast milk from central Taiwan and their relation to infant birth outcome and maternal menstruation effects. Environ Int. 2007, 33(2):239-45.
19. Main KM, Kiviranta H, Virtanen HE, Sundqvist E, Tuomisto JT, Tuomisto J, Vartiainen T, Skakkebćk NE, Toppari J. Flame retardants in placenta and breast milk and cryptorchidism in newborn boys. Environ Health Perspect. 2007, doi:10.1289/ehp.9924 (available at http://dx.doi.org/).
20. McDonald TA. Polybrominated diphenylether levels among United States residents: daily intake and risk of harm to the developing brain and reproductive organs. Integr Environ Assess Manag. 2005, 1(4):343-54.
21. Bromide Science and Environmental Forum (BSEF). Home Page [web page]. Brussels, Belgium: Bromide Science and Environmental Forum (BSEF), 2007. Available at http://www.bsef.com/. Accessed 19 Apr 2007.
22. Harrad S, Diamond M. New Directions: Exposure to polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs): Current and future scenarios. Atmos Environ. 2006, 40(6):1187-8.

*Identifying Gaps in Breast Cancer Research*
23. Hunter R. Phasing out the use of polybrominated diphenyl ethers. Revised Code of Washington: Public Health and Safety: 2007 Apr 17; Engrossed Substitute House Bill 1024, 1-13.
24. Pingree, H. Brominated Flame Retardants in Consumer Products. Public Laws of Maine, Title 38, Chapter 16- D, Sections 1691-1692.
25. Alaee M, Arias P, Sjodin A, Bergman A. An overview of commercially used brominated flame retardants, their applications, their use patterns in different countries/regions and possible modes of release. Environ Int. 2003, 29(6):683-9.
26. Christensen JH, Platz J. Screening of polybrominated diphenyl ethers in blue mussels, marine and freshwater sediments in Denmark. J Environ Monit. 2001, 3(5):543-7.
27. Darnerud PO. Toxic effects of brominated flame retardants in man and in wildlife. Environ Int. 2003, 29(6):841-53.

28. Environmental Working Group (EWG). High Levels of PBDE Found in House Dust (Part 2). In: Environmental Working Group (EWG). In the Dust: Toxic Fire Retardants in American Homes. Washington, DC, USA: Environmental Working Group (EWG), 2004 . Available at http://www.ewg.org/reports/inthedust/part2.php.
29. Hale RC, Alaee M, Manchester-Neesvig JB, Stapleton HM, Ikonomou MG. Polybrominated diphenyl ether flame retardants in the North American environment. Environ Int. 2003, 29(6):771-9.
30. Hale RC, La Guardia MJ, Harvey EP, Gaylor MO, Mainor TM, Duff WH. Flame retardants. Persistent pollutants in land-applied sludges. Nature. 2001, 412(6843):140-1.
31. Rudel RA, Camann DE, Spengler JD, Korn LR, Brody JG. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. Environ Sci Technol. 2003, 37(20): 4543-53.
32. Stapleton HM, Dodder NG, Offenberg JH, Schantz MM, Wise SA. Polybrominated diphenyl ethers in house dust and clothes dryer lint. Environ Sci Technol. 2005, 39(4):925-31.
33. Strandberg B, Dodder NG, Basu I, Hites RA. Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air. Environ Sci Technol. 2001, 35(6):1078-83.
34. Harrad S, Hunter S. Concentrations of polybrominated diphenyl ethers in air and soil on a rural-urban transect across a major UK conurbation. Environ Sci Technol. 2006, 40(15):4548-53.

*California Breast Cancer Research Program*
35. Bradman A, Fenster L, Sjodin A, Jones RS, Patterson DG, Eskenazi B. Polybrominated diphenyl ether levels in the blood of pregnant women living in an agricultural community in California. Environ Health Perspect. 2006, doi:10.1289/ehp.8899 (available at http://dx.doi.org/).
36. Fischer D, Hooper K, Athanasiadou M, Athanassiadis I, Bergman A. Children show highest levels of polybrominated diphenyl ethers in a California family of four: a case study. Environ Health Perspect . 2006, 114(10):1581-4.
37. Guvenius DM, Aronsson A, Ekman-Ordeberg G, Bergman A, Noren K. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenylols, and pentachlorophenol. Environ Health Perspect. 2003, 111(9):1235-41.
38. Morland KB, Landrigan PJ, Sjodin A, Gobeille AK, Jones RS, McGahee EE, Needham LL, Patterson DG Jr. Body burdens of polybrominated diphenyl ethers among urban anglers. Environ Health Perspect. 2005, 113(12):1689-92.
39. Schecter A, Johnson-Welch S, Tung KC, Harris TR, Papke O, Rosen R. Polybrominated diphenyl ether (PBDE) levels in livers of U.S. human fetuses and newborns. J Toxicol Environ Health A. 2007, 70(1):1-6.
40. Schecter A, Pavuk M, Papke O, Ryan JJ, Birnbaum L, Rosen R. Polybrominated diphenyl ethers (PBDEs) in U.S. mothers' milk. Environ Health Perspect. 2003, 111(14):1723-9.
41. Betts KS. A new record for PBDEs in people. Environ Sci Technol. 2005, 39(14):296A.
42. Johnson-Restrepo B, Kannan K, Rapaport DP, Rodan BD. Polybrominated diphenyl ethers and polychlorinated biphenyls in human adipose tissue from New York. Environ Sci Technol. 2005, 39(14):5177-82.
43. Allen JG, McClean MD, Stapleton HM, Nelson JW, Webster TF. Personal exposure to polybrominated diphenyl ethers (PBDEs) in residential indoor air. Environ Sci Technol. 2007, 41(13):4574-9.
44. Schecter A, Papke O, Harris TR, Tung KC, Musumba A, Olson J, Birnbaum L. Polybrominated diphenyl ether (PBDE) levels in an expanded market basket survey of U.S. food and estimated PBDE dietary intake by age and sex. Environ Health Perspect. 2006, 114(10):1515-20.
45. Betts KS. Finding PBDEs in couches and TVs: Researchers are using X-ray fluorescence to pinpoint household goods that are responsible for PBDEs in indoor air and dust [article]. In: Environmental Science & Technology: Online News. Washington, DC, USA: American Chemical Society, 2007 Jun 13. Available at http://pubs.acs.org/subscribe/journals/esthag-w/2007/june/science/kb_pbde.html.

46. Betts KS. The risk of PBDEs in dust. Environ Sci Technol. 2007, 41(5):1505-6.

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47. Wu N, Herrmann T, Paepke O, Tickner J, Hale R, Harvey LE, La Guardia M, McClean MD, Webster TF. Human exposure to PBDEs: associations of PBDE body burdens with food consumption and house dust concentrations. Environ Sci Technol. 2007, 41(5):1584-9.
48. Eriksson J, Green N, Marsh G, Bergman A. Photochemical decomposition of 15 polybrominated diphenyl ether congeners in methanol/water. Environ Sci Technol. 2004, 38(11):3119-25.
49. Gerecke AC, Hartmann PC, Heeb NV, Kohler HP, Giger W, Schmid P, Zennegg M, Kohler M. Anaerobic degradation of decabromodiphenyl ether. Environ Sci Technol. 2005, 39(4 ):1078-83.
50. He J, Robrock KR, Alvarez-Cohen L. Microbial reductive debromination of polybrominated diphenyl ethers (PBDEs). Environ Sci Technol. 2006, 40(14):4429-34.
51. Stapleton HM, Alaee M, Letcher RJ, Baker JE. Debromination of the flame retardant decabromodiphenyl ether by juvenile carp (Cyprinus carpio) following dietary exposure. Environ Sci Technol. 2004, 38(1):112-9.
52. Stapleton HM, Brazil B, Holbrook RD, Mitchelmore CL, Benedict R, Konstantinov A, Potter D. In vivo and in vitro debromination of decabromodiphenyl ether (BDE 209) by juvenile rainbow trout and common carp. Environ Sci Technol. 2006, 40(15):4653-8.
53. Huwe JK, Smith DJ. Accumulation, whole-body depletion, and debromination of decabromodiphenyl ether in male sprague-dawley rats following dietary exposure . Environ Sci Technol. 2007, 41(7):2371-7.
54. Sjodin A, Carlsson H, Thuresson K, Sjolin S, Bergman A, Ostman C. Flame retardants in indoor air at an electronics recycling plant and at other work environments. Environ Sci Technol. 2001, 35(3):448-54.
55. Thomsen C, Lundanes E, Becher G. Brominated flame retardants in archived serum samples from Norway: a study on temporal trends and the role of age. Environ Sci Technol. 2002, 36(7):1414-8.
56. Sjodin A, Hagmar L, Klasson-Wehler E, Kronholm-Diab K, Jakobsson E, Bergman A. Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers. Environ Health Perspect. 1999, 107(8):643-8.
57. Thuresson K, Bergman A, Jakobsson K. Occupational exposure to commercial decabromodiphenyl ether in workers manufacturing or handling flame-retarded rubber. Environ Sci Technol. 2005, 39(7):1980-6.
58. Jones-Otazo HA, Clarke JP, Diamond ML, Archbold JA, Ferguson G, Harner T, Richardson GM, Ryan JJ, Wilford B. Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure to PBDEs. Environ Sci Technol. 2005, 39(14):5121-30.

*California Breast Cancer Research Program*
59. Noren K, Meironyte D. Contaminants in Swedish human milk. Decreasing levels of organochlorine and increasing levels of organobromine compounds. Organohalogen Compounds. 1998, 38:1-4.
60. Meerts IA, van Zanden JJ, Luijks EA, van Leeuwen-Bol I, Marsh G, Jakobsson E, Bergman A, Brouwer A. Potent competitive interactions of some brominated flame retardants and related compounds with human transthyretin in vitro. Toxicol Sci. 2000, 56(1):95-104.
61. Zhou T, Taylor MM, DeVito MJ, Crofton KM. Developmental exposure to brominated diphenyl ethers results in thyroid hormone disruption. Toxicol Sci. 2002, 66(1):105-16.
62. Harju M, Hamers T, Kamstra JH, Sonneveld E, Boon JP, Tysklind M, Andersson PL. Quantitative structure- activity relationship modeling on in vitro endocrine effects and metabolic stability involving 26 selected brominated flame retardants. Environ Toxicol Chem. 2007, 26(4):816-26.

63. Kester MH, Bulduk S, van Toor H, Tibboel D, Meinl W, Glatt H, Falany CN, Coughtrie MW, Schuur AG, Brouwer A, Visser TJ. Potent inhibition of estrogen sulfotransferase by hydroxylated metabolites of polyhalogenated aromatic hydrocarbons reveals alternative mechanism for estrogenic activity of endocrine disrupters. J Clin Endocrinol Metab. 2002, 87(3):1142-50.
64. Meerts IA, Letcher RJ, Hoving S, Marsh G, Bergman A, Lemmen JG, van der Burg B, Brouwer A. In vitro estrogenicity of polybrominated diphenyl ethers, hydroxylated PDBEs, and polybrominated bisphenol A compounds. Environ Health Perspect. 2001, 109(4):399-407.
65. Olsen CM, Meussen-Elholm ET, Holme JA, Hongslo JK. Brominated phenols: characterization of estrogen-like activity in the human breast cancer cell-line MCF-7. Toxicol Lett. 2002, 129(1-2):55-63.
66. Stoker TE, Cooper RL, Lambright CS, Wilson VS, Furr J, Gray LE. In vivo and in vitro anti-androgenic effects of DE-71, a commercial polybrominated diphenyl ether (PBDE) mixture. Toxicol Appl Pharmacol . 2005, 207(1):78-88.
67. Canton RF, Sanderson JT, Nijmeijer S, Bergman A, Letcher RJ, van den Berg M. In vitro effects of brominated flame retardants and metabolites on CYP17 catalytic activity: a novel mechanism of action? Toxicol Appl Pharmacol. 2006, 216(2):274-81.
68. United States Environmental Protection Agency (US EPA), Office of Research and Development. Integrated Risk Inforamtion System (IRIS): Decabromodiphenyl ether (DBDPE) CASRN 1163-19-5) [web page]. Atlanta, GA, USA: United States Environmental Protection Agency (US EPA), 1995. Available at http://www.epa.gov/iris/subst/0035.htm. Accessed 28 Feb 2007.
69. Turken O, Narin Y, Demirbas S, Onde ME, Sayan O, Kandemir EG, Yaylaci M, Ozturk A. Breast cancer in association with thyroid disorders. Breast Cancer Res. 2003, 5(5):R110-3.


70. Talsness CE, Shakibaei M, Kuriyama SN, Grande SW, Sterner-Kock A, Schnitker P, de Souza C, Grote K, Chahoud I. Ultrastructural changes observed in rat ovaries following in utero and lactational exposure to low doses of a polybrominated flame retardant. Toxicol Lett. 2005, 157(3):189-202.
71. Kuriyama SN, Talsness CE, Grote K, Chahoud I. Developmental exposure to low dose PBDE 99: effects on male fertility and neurobehavior in rat offspring. Environ Health Perspect. 2005, 113(2):149-54.
72. Laws S, Ferrell J, Hedge J, Crofton K, Cooper R, Stoker T. The effects of DE-71, a commercial polybrominated diphenyl ether mixture, on female pubertal development and thyroid function. [abstract]Toxicologist. 2003, 72:137.
73. Lichtensteiger W, Ceccatelli R, Faass O, Fleischmann I, Schlumph M. Effects of polybrominated diphenylether (PBDE) on reproductive organ and brain development and gene expression in rats. [abstract]Toxicologist. 2003, 72:133.
74. Stoker TE, Laws SC, Crofton KM, Hedge JM, Ferrell JM, Cooper RL. Assessment of DE-71, a commercial polybrominated diphenyl ether (PBDE) mixture, in the EDSP male and female pubertal protocols. Toxicol Sci. 2004, 78(1):144-55.
75. Hardell L, Lindstrom G, van Bavel B, Wingfors H, Sundelin E, Liljegren G. Concentrations of the flame retardant 2,2',4,4'-tetrabrominated diphenyl ether in human adipose tissue in Swedish persons and the risk for non-Hodgkin's lymphoma. Oncol Res. 1998, 10(8):429-32.
76. Hardell L, Bavel B, Lindstrom G, Eriksson M, Carlberg M. In utero exposure to persistent organic pollutants in relation to testicular cancer risk. Int J Androl. 2006, 29(1):228-34.

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**Testimony of Pamela Miller, Executive Director, Alaska Community Action on Toxics**

**For a Hearing of the Alaska State House Labor and Commerce Committee Concerning House Bill 63—An Act Relating to Flame Retardants and Other Bioaccumulative Toxic Chemicals**

**March 14, 2011**

I would like to begin by thanking Chairman Olson and Members of House Labor and Commerce Committee for this opportunity to provide testimony in support of HB 63. My name is Pamela Miller, Biologist and Executive Director of Alaska Community Action on Toxics (ACAT). ACAT is a statewide environmental health organization that conducts research and provides educational programs, technical assistance, and training.

HB 63 is an important measure to protect public health—especially the health of vulnerable populations, including children as well as firefighters who are on the front lines of exposure to these dangerous chemicals. Polybrominated diphenyl ethers or PBDEs are similar in structure to the banned chemicals known as PCBs, polychlorinated biphenyls, and thus can have similar harmful effects on the body. PBDEs accumulate and are long-lasting, and we are concerned about them because they interfere with proper thyroid function in laboratory studies, cause problems with brain development and disrupt learning, memory, and behavior.

Over the past year, new scientific evidence has emerged about the health effects of brominated flame retardants that compels

urgent action. Dr. Julie Herbstman of the Columbia University Center for Children's Environmental Health found that children who had higher prenatal exposures to PBDEs scored lower on tests of mental and physical development at ages 1, 4, and 6. Developing children are exposed to these toxic chemicals before they are born and this affects their learning and development throughout childhood and perhaps permanently. Concentrations of cord blood PBDEs in this study are similar to developing children throughout the U.S.

As with public policy measures to reduce childhood exposure to lead, actions that we take now to reduce and eliminate exposure to PBDEs will have immeasurable benefits in preventing intellectual deficits in generations of children. You may know of the devastating effects of lead on the intellectual capacity of generations of children because it was used as an additive in leaded gasoline and paint. There were dramatic public health benefits for our children's intellectual capacity as well as immense savings in health care costs as lead was removed from paint and gasoline. We didn't stop painting our homes or driving our cars, we removed the lead in favor of safer alternatives.

By updating our state laws governing toxic chemicals, we can improve the health of Alaska's citizens and contain health care costs. The federal system for regulating chemicals is broken and states are becoming the leaders in developing innovative laws that protect public health—based on the common sense principle that hazardous or untested chemicals do not belong in consumer products. We support the aspect of this legislation to list persistent bioaccumulative toxics (or PBT chemicals) because it provides the public with important, transparent right-to-know provisions about other harmful chemicals—something that Alaskans value highly. We also support the definition for PBTS provided in SB 27 and as adopted in the committee substitute.

Chemicals policy reform is good for business. Leading companies are highly motivated to identify and use safer alternatives—consumers are demanding it. Today's business leaders are

concerned about the health and business impacts that could arise if their products contain toxic chemicals. There are new opportunities for businesses to innovate and make US businesses more competitive in the global marketplace and in creating new jobs. Several states, including Michigan, New York, Maine, and California are investing in green chemistry research and development as an emerging source of new jobs and economic development.

I know health care cost containment is a high priority for this legislature. Knowing this, I hope that this information will be useful. Growing scientific evidence demonstrates that some chemicals widely used in consumer products contribute to an epidemic of chronic diseases and disorders. According to the U.S. Centers for Disease Control, chronic diseases now account for 70% of deaths and 75% of health care costs. While PBDEs and other toxic chemicals alone are not responsible for all of these health outcomes, we know for example: 1) learning and developmental disabilities affect one in six children in the U.S. ; 2) about 12% of women reported difficulty in conceiving and maintaining pregnancy, an increase of 40% since 1982; 3) as much as 90% of childhood cancers may be explained by environmental exposures based on a review of 31 studies of childhood cancer. Male reproductive health has declined as evidence of decreased sperm count and quality, birth defects of the genitals, and 60% increase in testicular cancer between 1973 and 2003. We have an ethical responsibility as a society to protect health and reduce the costs of health care by preventing unnecessary exposures to chemicals that are known to cause harm, including PBDEs. Children have a right to live in an environment in which they can reach and maintain their full potential.

People are exposed to PBDEs through contaminated air, household dust, and foods. These chemicals leach out of products and we are exposed through indoor air and dust. PBDEs harm childhood brain development, and can contribute to learning disabilities that last into adulthood. Exposure to deca- and its

breakdown products can harm women and children because they are passed to infants through breast milk and to children through contact with household dust. Levels in wildlife and people are increasing exponentially. PBDEs in human breast milk are increasing and doubling every 2-5 years. Levels in the U.S. are the highest of all countries for which there are data and about 10-100 times greater than human tissue levels in Europe. PBDEs are persistent and can travel long distances via wind and ocean currents—PBDEs are now ubiquitous and found in northern air, sediments and wildlife. Alaskans are more vulnerable to exposures due to our higher levels of consumption of fish and marine mammals. Women of child-bearing age in the Yukon-Kuskokwim region of Alaska have the highest levels of any human population in the circumpolar Arctic.

Also in the past year, approximately 200 prominent scientists signed a consensus statement known as the San Antonio Statement on Brominated and Chlorinated Flame Retardants published in the December 2010 issue of Environmental Health Perspectives (included in your bill packet), the peer-reviewed journal of the National Institute of Environmental Health Sciences. The statement names brominated flame retardants as a class of substance of concern for persistence, bioaccumulation, long-range transport and toxicity. Among the twenty points, the scientists state: ***"Brominated and chlorinated flame retardants can increase fire toxicity, but their overall benefit in improving fire safety has not been proven."***

The American Public Health Association recognizes the public health threat presented by the prevalence of toxic PBDE flame retardants and passed a resolution stating: *"In light of the emerging science on the inherent toxicity and persistence of PBDEs, evidence of adverse health effects on animals, and the prevalence and rising levels in fish, biota, and human breast milk, immediate action is needed to prevent further environmental contamination and to protect public health. The American Public Health Association urges state and federal governments to*

*require the use of all PBDE flame retardants be phased out in all products manufactured and sold in the U.S. by a date certain."*

No one disputes the importance of fire prevention and safety, however, with the availability and economic viability of safer alternatives, it does not make sense to expose people to these toxic chemicals. In summary, we urge you to pass HB 63 in order to protect health, particularly the health of vulnerable populations including pregnant women, developing children, fire fighters and other workers. Thank you.